CHAPTER-I

INTRODUCTION
Sleep is not a simple process. Many different parts of the brain control and influence sleep at different stages. Getting a good night's sleep is essential for feeling refreshed and alert during the day. Unfortunately, not everyone is able to get the restorative sleep they need. A sleep disorder (somnipathy) from a clinical point of view is, “a disruptive pattern of sleep that may include difficulty falling or staying asleep, falling asleep at inappropriate times, excessive total sleep time, or abnormal behaviors associated with sleep (Czeiler, Weitzman et al, 1980; Doghramji and Paul, 2004). There may be two natural daily peak times for sleeping: at night and at mid-day (Bonnet and Webb, 1979), usually known as night sleep and afternoon nap. What follows bears with night sleep only.

There are two distinct phases of sleep:

- **Non-rapid eye movement (NREM) sleep** - The quiet or restful phase of sleep, also referred to as "slow wave sleep"; it is divided into four stages of progressively deepening sleep
- **Rapid eye movement (REM) sleep** - The phase of sleep in which the brain is active and dreaming occurs; it is also known as "dream sleep".

**NATURAL HISTORY OF SLEEP**

Sleep is not simply the absence of wakefulness. Rather, it is a complex state of active and coordinated brain processes. As demonstrated by electroencephalography, sleep progresses in four deepening stages, plus rapid-eye-movement (REM) sleep. The four stages are collectively known as non-REM sleep. Stage 1 is a transitional "drowsy" phase that precedes deeper, more restorative stages. Relative to non-REM sleep, REM sleep is associated with brain activity that resembles wakefulness and is linked to most recallable dreams.

First, let us take a closer look at the measured stages of sleep as they have been put forth by sleep research scientists. After the first stage has ended, which is a transitional stage between waking and sleeping, the second stage, light sleep, begins. Many researchers see this second stage as the actual beginning of sleep. The third and fourth stages are often referred to together as the stage of deep-sleep. After a renewed transitional phase between waking and sleeping, there follows a completely new sleep quality, the so-called REM phase (dream-phase). Then a new cycle begins.
In one night, this cycle is repeated four or five times. The deepest sleep phases occur in the first two sleep cycles. In contrast, the REM phases become longer with each succeeding cycle. Stages one through four (non-REM sleep) make up approximately seventy to eighty percent of the entire sleeping period; REM sleep accounts for about twenty to thirty percent. Approximately every ninety minutes a REM phase occurs which lasts twenty minutes on average but increases in length throughout the night. Deep sleep and REM sleep demonstrate a counteractive duration. After the first stage of falling asleep, sleep is deeper and therefore it is more difficult to awaken a person. Physical regeneration occurs mainly during the first stage. Towards the end of the night, dreams and soul-spiritual regeneration are at the forefront. One additional note: REM sleep makes up a larger proportion of total sleep time for children than for adults.

The total amount and composition of sleep change throughout life. With aging, the total amount of sleep shortens: infants and children normally sleep 16 to 20 hours a day, adults sleep seven to eight hours a day and, after age 60, adults sleep approximately 6.5 hours a day (Feinberg, Koreisko and Heller, 1967). Delta sleep (stages 3 and 4 sleep), the deepest and most refreshing kind of sleep, diminishes markedly with age. In contrast, early stage 1 sleep, the lightest sleep, increases with age. The threshold for arousal is lowest during stage 1 sleep and highest during delta sleep, a feature that helps explain why sleep in old age becomes more fragmented, with more brief awakenings (Kales and Kales, 1974). There is little decline in REM sleep throughout a person's lifetime.

Research suggests that Non REM sleep may play a role in bolstering the immune system and may also be tied to the rhythms of the digestive system (Yamadara, Takahshi and Okawea, 1996). There are 24-hour body rhythms known as circadian rhythms including changes in body temperature, plasma levels of various hormones and variables such as heart rate and blood pressure. These and many other variables increase and decrease at various times during the day and night and determine when we need to sleep and be most alert (Edgar, 1996).

Since the present proposal focuses upon chronic insomnia, other sleep problems shall not be detailed.
DEFINITION OF INSOMNIA

Insomnia, or agrypnia, is the lack of sleep at times when convention dictates that one should be asleep. The definition of insomnia is that it is a condition wherein a person has problems regarding his/her patterns of sleep. Falling asleep, being unable to remain asleep are examples of disrupted sleep patterns. Some insomniacs have trouble falling asleep and remaining asleep. The subject must also suffer some degree of impairment in social, occupational, or other important areas of daytime functioning (Carskadon and Dement, 1977).

The International Classification of Sleep Disorders (DSM, 1997) defines insomnia as, “difficulty initiating and/or maintaining sleep.” Many variations on this definition have been proposed or used in research, and a consensus definition of insomnia has not been reached. National Institutes of Health in 2005 stated, “insomnia is an experience of inadequate or poor quality sleep characterized by one or more of the following: difficulty falling asleep, difficulty maintaining sleep, waking up too early in the morning, non-refreshing sleep.” Insomnia also involves daytime consequences, such as “tiredness, lack of energy, difficulty concentrating, and irritability.” Insomnia lasting less than 4 weeks is considered to be transient. Insomnia lasting longer than 4 weeks is chronic.

Thus, insomnia is a common, final pathway where a variety of factors converge. Insomnia can be transient or chronic, primary or secondary, somatic or psychogenic, extrinsic or intrinsic, etc.

DIAGNOSIS

“Insomnia” is used to refer to both a symptom and a disorder. The symptom of insomnia is defined as a subjective Complaint of difficulty falling asleep, difficulty staying asleep or poor quality sleep. In DSM-IV-TR (American Psychiatric Association, 2000), insomnia symptoms are included among the diagnostic criteria for several other mental disorders, including major depressive disorder and generalized anxiety disorder. Insomnia disorders are characterized by insomnia symptoms accompanied by significant distress or impairment. In DSMIV- TR the specific diagnosis of primary insomnia is further defined by duration of at least 1 month and by symptoms that do not occur exclusively during the course of another sleep disorder, mental
disorder, or medical disorder or result from use of substances or medications. Finally, DSMIV-TR includes diagnoses of “secondary” insomnia disorders, that is, insomnia that causes significant distress or impairment or warrants independent clinical attention but is believed to be directly related to a coexisting mental disorder or medical disorder or to the effects of substances or medications.

Sleep medicine specialists use the International Classification of Sleep Disorders, Second Edition (ICSD-2) (American Academy of Sleep Medicine, 2005), to diagnose sleep disorders. ICSD-2 describes general criteria that are common to all insomnia disorders as well as eight specific insomnia disorders (in addition to “unspecified” and “not otherwise specified” categories), each of which meets the general criteria along with more specific diagnostic criteria. For most purposes, the general insomnia criteria proposed in ICSD-2 serve as a useful basis for discussing insomnia as a clinical disorder. Other classification schemes for insomnia have also been used. For instance, symptom-based classifications distinguish between sleep-onset and sleep-maintenance insomnia. However, longitudinal studies suggest that these specific symptoms have limited stability over time (Hohagen, Kappler et al, 1994); patients who present with sleep-onset insomnia at one time may present with frequent awakenings at a later time. Moreover, a majority of patients present with some combination of symptoms.

Duration-based classifications (acute, short-term, and chronic) have also been suggested (National Institutes of Health, 1984). Such classifications may provide clues to the cause of insomnia. For instance, acute and short-term insomnia are more often related to life stresses, acute illnesses, or medications, whereas chronic insomnia is more likely to be related to behavioral factors or the effects of chronic mental or medical disorders. However, the majority of patients in clinical trials and in clinical practice have chronic symptoms, which is the focus of this review. Distinctions have also been drawn between primary and secondary insomnia disorders. The underlying rationale is that secondary insomnia is caused by another disorder, whereas primary insomnia has no other identifiable cause. However, as noted in the 2005 National Institutes of Health (NIH) State of the Science Conference statement on the Manifestations and Management of Chronic Insomnia in Adults, such distinctions may not be helpful clinically. Insomnia often has multiple causes, and distinguishing when another condition “causes” insomnia can be difficult. For instance, the course of insomnia may not follow the
course of the other disorder, and the two conditions may require different treatments. For this reason, the NIH conference commended the term “comorbid insomnia” as a preferable alternative to the term “secondary insomnia.”

**EPIDEMIOLOGY**

**Prevalence and demographics**
Insomnia is the most common disorder of sleep (Bixler, Kales and Soldatos, 1979). The prevalence of insomnia differs depending on the specific case definition used and the population assessed. However, whatever definition is used, insomnia remains the most prevalent sleep disorder in the population. Insomnia symptoms—that is, the complaint in the absence of specific duration or distress criteria—occur in some 30%–40% of adults, and specific insomnia disorders—that is, the complaint together with meeting duration and impairment criteria—occur in 5%–10% of adults (Ohayon, 2002). The World Health Organization Collaborative Study which was conducted in 14 countries found that 15% of general health care attendees had a period of 2 weeks or longer when nearly every night they had trouble falling asleep or staying asleep. Rates of insomnia are higher in medical and psychiatric care settings than in the general population. A number of consistent risk factors for insomnia have been identified (Ohayon, 1997; Buysse, German et al, 2005). The strongest of these is concurrent depressive symptoms. Female sex is also consistently associated with insomnia, with a ratio of approximately 1.4:1. Increasing age, comorbid medical disorders, and comorbid psychiatric disorders are other consistent risk factors.

A number of surveys have shown that insomnia occurs more frequently with increasing age (Bixler, Kales et al, 1979; Johns, Egan et al, 1970; Karacan, Thornby et al, 1978; McGhee and Russell, 1962; Tune, 1968; Weiss, Kasinoff & Bailey, 1962; Selected Symptoms of Psychological Distress, 1970) and in women (Hammond, 1964; Karakan, Bonnet et al, 1980; McGhee and Russell, 1962; Tune, 1968; Selected Symptoms of Psychological Distress, 1970). As women become older they complain more of difficulty falling asleep as well as reporting lighter sleep with more frequent awakenings (The Gallup Organization, 1979). An increased prevalence of insomnia has also been associated with psychological disturbances and lower
socioeconomic status (Karacan, Thornby et al, 1978; McGhee and Russell, 1962; Tune, 1968; Simonds and Parraga, 1982). These two factors that increase the likelihood of insomniac complaints appear to be related, because mental health disorders are more prevalent among persons of lower socioeconomic status (Hollingshead and Redlich, 1958; Srole, Langer et al, 1962), and social class have been found to be inversely related to degree of life stress, as measured by life change events (Dohrenwend, 1970).

Longitudinal studies suggest that approximately 50% of individuals with insomnia continue to have symptoms after follow up periods of 1 year or longer and most cross-sectional studies of insomnia patients report a duration of several years (Buysse, Angst et al, 2008; Foley, Monjan et al, 1999; Moul, Nofzinger et al, 2002). Although the number of true longitudinal insomnia studies is small, evidence suggests that improvement in medical and psychiatric conditions is associated with improvement in insomnia (Katz and McHorney, 1998). As noted above, cross-sectional associations between insomnia and psychiatric disorders are strong, but additional evidence suggests that insomnia is a risk factor for the development of psychiatric disorders and for poor outcomes in these disorders. For instance, approximately a dozen longitudinal studies have demonstrated that insomnia is an independent risk factor for subsequent development of depression (Perlis, Smith et al, 2006). This relationship has been observed from adolescence to later adulthood and is maintained after adjustment for concurrent depressive symptoms. In addition, insomnia is one of the most common persistent symptoms in individuals treated for depression, and its presence is a risk factor for no response to depression treatment and for recurrence following remission (Buysse, Cherry et al, 1999; Nierenberg, Keefe et al, 1999; Reynolds, Frank et al, 1997).

We conclude from these investigations that a reasonable prevalence estimate for chronic insomnia about 10%. When considering insomnia of any duration or severity, between 30% and 50% of the general population appears to be affected.

**CAUSES**

Decades of scientific research have shown that sleepiness and fatigue, as well as sleeplessness, affect everyone's health, safety, productivity and well-being; Bootzin and Engel (1981); Taylor,
Daniel et al (2003). Yet most people are slow to seek the advice and treatment of a healthcare provider for sleep problems and excessive sleepiness.

Although some risk factors and etiologies of insomnia have been identified, the nature of the relationships has not been fully elucidated. Some risk factors for insomnia that have emerged from data related to insomnia include female gender (Li, Wing & Fong, 2002) and old age (Jean-Louis, Magai et al, 2001) Additional risks factors include less education, unemployment, separation or divorce, and medical illness (Silber, 2005; Hajak, 2001) Insomnia may be primary or secondary to other sleep problems and may be associated with a number of co-morbidities. An association has been found between insomnia and psychiatric (depression and anxiety) and psychological disorders (Klink, Quan et al, 1992).

Environmental factors such as irregular sleep schedules, use of caffeine or other stimulants, co-morbid medical conditions, and/or shift work may also predispose vulnerable individuals to insomnia.

Insomnia can originate by any of a number of factors such as physical illness, a stress-filled lifestyle, excessive caffeine consumption, or chronic pain. It may simply be the result of poor sleeping habits, such as napping during the day and going to bed at irregular hours. Insomnia can often be linked to alcohol or drug abuse and to misuse of certain medications. In addition, sleep patterns change considerably with age (Williams, Karacan & Frazier; 1978).

Chronic insomnia is believed to primarily occur in patients with predisposing or constitutional factors. These factors may cause the occasional night of poor sleep but not chronic insomnia. A precipitating factor, such as a major life event, causes the patient to have acute insomnia. If poor sleep habits or other perpetuating factors occur in the following weeks to months, chronic insomnia develops despite the removal of the precipitating factor.
CONSEQUENCES OF INSOMNIA

There is increasing evidence that chronic insomnia may predispose individuals to the development of psychiatric disorders (Chang, Ford et al, 1997; Hajak, 2001; Buysse, Angst et al, 2008). Persistent insomnia increases the risk of depression, substance abuse, and anxiety disorders.

Insomnia is a syndrome composed of symptoms of disturbed sleep, decrement of day time performance and depressed mood to various degrees and in various combinations (Bootzin and Engel, 1981). To understand the effects of insomnia or lack of adequate quality sleep, it is imperative to understand sleep. It is a very complicated process involving body and brain. When this process is incomplete, the body and brain are not fully fit for the next day’s activity. Insomnia is also a health risk factor as revealed in a study by Lichstein and Johnson (1993). The person feels very tired, fatigued, energy levels reduced and reflexes slowed. The sensitivity of the senses is blunted. Our mental functions like our ability to learn, remember and concentrate are affected by insomnia. The performance level in the workplace drops (Johnson and McLeod, 1973). The mind becomes sluggish and looses concentration. Lack of attention, dizzy spells and mood changes are other effects during the daytime caused by sleepless nights. Johnson and McLeod in 1973 also reported decrements in the area of intellectual functions and mood. Some people become very irritable and flare up for no reason. It may sometimes lead to emotional problems and depression (Reimann, Dieter et al, 2003). Relationship between depression and impairment of sleep quality is confirmed in a study conducted by De Gennaro, Luigi et al (2004).

In chronic cases, when suffering from severe insomnia and sleepless nights continue for long time, people can even experience hallucinations.

If a person suffers from insomnia, driving and operating machinery are very dangerous. Abnormal sleep duration is highly related to motor vehicle crash risk. (Marshall, Nathenial et al, 2004). Insomnia though a symptom of some underlying disorder, becomes a cause of other physical and psychological illness when it is allowed to go on for a long time (Horne, 1978). McGaugh, Jenson and Martinez (1979) gave references for the relevance of sleep for memory processes.
Anyone who has lived through a bout of insomnia knows the side effects of this condition can be horrific. The effects of insomnia can range in severity and can be based on the length of time the condition continues. Despite this, even the most basic of the effects of insomnia can put a real crimp in how a person goes through everyday life (Gluckman and Jason, 1994).

When the effects of insomnia over a long period of time are considered, it makes sense for people to seek out solutions. Whether the answer lies in stress reduction or medical attention, getting help can make a big difference. When sleep returns to normal, the effects of insomnia can be removed or at least lessened.

The effects of insomnia can include such things as personality changes, performance issues, psychological problems, problem with Safety and health. Finding what is causing the insomnia and dealing with it can help life return to normal. It might take a little time to get to the root cause, but doing so can be vital for a whole lot of reasons (Pesic and Milos, 2007). Either total sleep length (Williams, Karcan and Frazier, 1978) or delta sleep is regarded as restorative for the body whereas REM sleep is restorative for the brain (Vogel, 1975). Therefore, any decrement in sleep shall result into behavioral impairments.

Chronic insomnia is, in turn, a significant risk factor for the development of psychiatric illness. Victims of insomnia are more than twice as likely as no insomniacs to have psychiatric disorders and are more prone to subsequent depressive illness, anxiety, or alcohol-abuse.

Insomnia has significant direct and indirect effects on the health and wellness of affected individuals. Insomnia has been correlated with frequent use of medical services (Simon and VonKorff, 1997; Leger, Guilleminault and Bader, 2002), chronic health problems, (Ohayon, 1997; Roberts, Shama and Kaplion, 1999), increased drug use, and perceived poor health (Foley, Monjan et al, 1995), and has been associated with medical problems including heart disease (Janson, Lindberg and Gislason, 2001), hypertension (Gislason, Reynisdottir and Kristbjarnarson, 1993), and musculoskeletal problems (Janson, Lindberg and Gislason et al, 2001). The daytime consequences of chronic insomnia often include increased healthcare utilization, increased risk of depression (Riemann, Dieter et al 2003), poor memory, reduced concentration, poor work performance, and perceived or real risk of failure at work (Kryger, Roth and Dement, 2000). The economic implications of insomnia and associated morbidity have
been described. The direct costs of insomnia (insomnia treatments, healthcare services, hospital and nursing home care) are estimated to be nearly $14 billion (Walsh and Engelhardt, 1999; Walsh and James, 2004). The indirect costs of insomnia, such as time lost from work and loss of productivity, are estimated to be nearly $28 billion. A National Sleep Foundation survey found that lost productivity from insomnia alone was over $18 billion.

Age, performance and sleep deprivation have been found to be interrelated and have been discussed in terms of vulnerability to sleep related accidents (Philip, Pierre et al, 2004).

A psychiatric disorder, such as depression, is frequently a cause and a consequence of chronic insomnia, especially in the elderly. The effect of a chronic psychiatric illness often leads a patient to self-medicate, producing even more severe insomnia. Other psychiatric conditions that can cause insomnia include anxiety disorder, panic disorder, mania and acute psychosis.

Conditioned insomnia occurs when the act of going to bed triggers anxiety and the inability to go to sleep. In other words, the patient has been inadvertently trained to stay awake at bedtime. A clue to this disorder may be the patient's ability to readily fall asleep at times when he or she is not focusing on obtaining slumber (i.e., unscheduled naps).

A major cause of sleep disturbance in middle-aged women is the menopause-related "hot flush." Recent studies indicate that nearly every hot flush promotes an arousal from sleep (Woodward and Freedman, 1994). Insomnia may be a reason for instituting hormone replacement therapy (Scharf, McDannold et al, 1997).

Certain medical conditions, such as gastroesophageal reflux disease, chronic obstructive pulmonary disease, peptic ulcer disease, prostatic hypertrophy (resulting in overflow incontinence) and congestive heart failure with associated paroxysmal nocturnal dyspnea, frequently disturb sleep and may be interpreted by the patient as insomnia. Recurrent enuresis may disturb sleep and, in an adult, necessitates a genitourinary work-up for incontinence or urinary tract infection. Patients with chronic pain, such as that resulting from fibromyalgia, may have mood and cognitive disturbances in addition to insomnia and early-morning awakening (Harding, 1998).
Insomnia can also be caused by:

- Psychoactive drugs or stimulants, including certain medication, herbs, caffeine, cocaine, ephedrine, amphetamines, methylphenidate, MDMA, methamphetamine and modafinil,
- Hormone shifts such as those that precede menstruation and those during menopause (Naitoh, 1976),
- Psychological problems like fear, stress, anxiety, emotional or mental tension, work problems, financial stress, unsatisfactory sex life,
- Mental Disorders such as clinical depression, bipolar disorder, general anxiety disorder,
- Disturbances of the circadian rhythm, such as shift work and jet lag can cause an inability to sleep at some times of the day and excessive sleepiness at other times of the day,
- Certain neurological disorders, brain lesions, or a history of traumatic brain injury,
- Medical conditions such as Hyperthyroidism and Wilson's syndrome Chronic pain syndromes, Congestive heart failure, chronic obstructive pulmonary disease (COPD), Degenerative diseases, such as Alzheimer disease,
- Abuse of over-the-counter or prescription sleep aids can produce Rebound insomnia,
- Poor sleep hygiene,
- **Parasomnia**, which includes a number of disruptive sleep events including nightmares, sleepwalking, violent behavior while sleeping, and REM behavior disorder, in which a person moves his/her physical body in response to events within his/her dreams,
- a rare genetic condition can cause a prior-based, permanent and eventually fatal form of insomnia called **fatal familial insomnia** and
- Sleep problems are usually a biochemical problem. Biochemical breakdown can take place in many ways. For example, if our digestive system is stressed and unable to digest protein, the amino acids, which affect neurotransmission, will not be available to our brain.
A wide range of disorders should be considered in the search for an underlying cause of chronic insomnia. Several etiologies may exist at the same time. Insomnia may be the effect of prescription or over-the-counter medications (Bethesda, 1984), or of a medical condition (Moran and Stoudemire, 1992). In addition, insomnia may represent a prodromal indication of psychiatric illness (particularly depression) (Eaton, Badawi and Melton, 1995), a sleep-related breathing disorder such as sleep apnea (Rajagopal, Abrecht et al, 1984; National Heart, lung and blood institute working on sleep apnea, 1996), a movement-related disorder such as restless leg syndrome (Ancoli-Israel, Kripke et al, 1991; Lin, Kaplan et al, 1998) or a circadian rhythm disorder.

Evaluation can be done by any or all of the following methods:

**Initial sleep history:**
A thorough clinical history (clinical interview and sleep history) is the cornerstone of evaluation for chronic insomnia (Buysse, Ancoli-Israel and Edinger, 2006). The evaluation should focus on the description of current symptoms, including not only the type of sleep disturbance at night but also sleep habits and patterns. The majority of insomnias are secondary to a medical, psychiatric, or behavioral disorder (Nowell, Buysse and Reynold, 1997); therefore, the investigator must inquire carefully and consider thoroughly the possible causes. In particular, the clinician should inquire about times of going to bed and getting out of bed, variability in sleep timing from day to day, and emotional, cognitive, and physical states surrounding sleep. Symptoms of other specific sleep disorders should also be considered. These include loud snoring and witnessed breathing pauses, which might suggest sleep apnea, and motor restlessness and involuntary leg movements, which might suggest restless legs syndrome. Daytime consequences associated with insomnia should also be evaluated. The most common complaints include mood disturbance (typically described as irritability and mood liability, rather than depression or anxiety), fatigue, and complaints of cognitive inefficiency or difficulty concentrating. The majority of patients with chronic insomnia do not actually complain of daytime sleepiness, that is, the tendency to fall
asleep in inappropriate situations. Rather, insomnia appears to be associated with difficulty sleeping at any time during the 24-hour day. The evaluation of insomnia should also include careful consideration of comorbid psychiatric and medical disorders as well as medications and substances that might interfere with sleep. Virtually any psychiatric disorder can be associated with insomnia, but depressive and anxiety disorders are especially common. Likewise, a wide range of medical and neurologic conditions can be associated with insomnia (Mahowald, Mahowald and Bundlie, 1989). Particular attention should be given to those conditions that are associated with pain, breathing difficulty, and impaired mobility. Medications that affect any CNS neurotransmitter can be associated with insomnia. Common examples include high doses of caffeine, alcohol, and antidepressants, particularly selective serotonin reuptake inhibitors, serotonin-norepinephrine reuptake inhibitors, and atypical antidepressants. It is often useful to view the insomnia history in terms of predisposing, precipitating, and perpetuating factors (Akpinar, 1987). These factors can help identify potential causes of insomnia and treatment targets. Predisposing factors include family/genetic diathesis and chronic mental or medical disorders that increase the likelihood of insomnia symptoms. Precipitating factors are acute events or experiences that may push vulnerable individuals over the threshold into acute insomnia. Perpetuating factors include behavioral patterns (e.g., spending excessive time in bed) and medical or medication factors that maintain insomnia symptoms over time.

**Questionnaires and sleep diary:**
The sleep clinical history can be usefully supplemented by collection of a 2-week sleep-wake diary. The collections of the patient’s sleep diary are mandatory when evaluating and treating insomnia (Buysse, Reynold and Kupfer, 1994; Morin, Kulbert and Schwartz, 1994). This is a prospective charting of a patient’s actual sleep hours and habits, and it can usefully identify variability in sleep patterns and specific daytime correlates that may provide targets for subsequent intervention.
Psychometric evaluation:
Psychological testing is used as a screening tool or when indicated for more extensive evaluation of neuropsychological symptoms in the patient with insomnia (Zorick, Roth and Hartse, 1981). For this, MMPI, mood scales, etc are chosen.

Physiological monitoring:
Specific laboratory testing has limited utility in the diagnosis and assessment of insomnia. However, in specific situations, general metabolic panels, CBC, or endocrine testing (e.g., thyroid hormone) may be useful. Overnight sleep studies with polysomnography are not routinely indicated in the evaluation of chronic insomnia (Sadeh, Hauri & Kripke, 1995; American Sleep Disorders Standards of Practice Committee, 1995). However, in specific situations, such testing may be useful. For example, individuals with a high index of suspicion for sleep apnea should be referred for polysomnographic testing. Risk factors include obesity, loud snoring, witnessed pauses in breathing, craniofacial abnormalities, or factors that might lead to a central dysregulation of breathing, such as congestive heart failure and stroke. Insomnia patients with unusual behaviors during sleep, such as violent behavior, should also be considered for polysomnography. Finally, failure to respond to usual insomnia treatments may warrant referral to a sleep specialist and, in some cases, polysomnography.

MANAGEMENT AND TREATMENT OF INSOMNIA
The causes of insomnia are many; likewise, the rational treatment plan is varied. The sleep specialist must explain the treatment to the patient and assess the patient’s understanding, motivation, and ability to participate in the plan. Management of chronic insomnia begins with attempts to identify and treat any underlying causes. There may be more than one cause of insomnia, but the causes may be difficult to identify. Drug therapy may be beneficial for short-term improvement, while behavioral intervention provides more sustained effects. Long-term use of many psychotropic or sedative-hypnotic drugs can cause adverse reactions and may actually impair sleep. Behavioral intervention combined with pharmacologic agents may be more effective than either approach alone.
**Pharmacological treatment**

Management of acute insomnia has traditionally involved pharmacotherapy. This is used for chronic insomnia only if non-pharmacologic approaches have been exhausted or as a complement to these treatments.

Pharmacologic management of insomnia is frequently prescribed and is efficient (Walsh, Roehrs and Roth, 2005). Antidepressant medications are used when the insomnia is associated with mood disorders (Karacan, Thornby et al, 1978). The use of such agents is common practice for both acute and chronic insomnia, despite the fact that the Food and Drug Administration (FDA) has approved none of them for chronic insomnia. Another medication, eszopiclone (Lunesta), was recently approved by the FDA for treatment of insomnia, but the duration of use is not explicitly stated. An estimated 0.5 percent of the population takes sedative medications for insomnia for more than 1 year (American Academy of Sleep Medicine, 2005). More than 1 in 10 people (11 percent) report using prescription (6 percent) and/or over-the-counter (OTC) medications (6 percent) at least a few nights a month, to help them sleep, according to a Sleep in America Poll. Individuals reporting symptoms of medical conditions are more likely to take sleep aids, both prescription and OTC medications. For example, 14 percent of people with symptoms of depression report using prescription medication, and 12 percent of people with symptoms of depression report using OTC sleep aids. Medications commonly used to treat insomnia include sedating antidepressants (Jacobs, Gregg et al, 2004), antihistamines, anticholinergics, benzodiazepines, and non-benzodiazepine hypnotics. A side effect of all hypnotics is to reduce slow wave sleep. Other side effects of concern are possible daytime residual effects related to sedation, rebound insomnia, and tolerance, along with minor side effects specific to each drug class. Many questions and challenges related to pharmacological therapy for chronic insomnia remains, such as the appropriate treatment for different types of primary and secondary insomnia, and the long-term side effects and daytime consequences of pharmacotherapy. The evidence for management of chronic insomnia with pharmacotherapy has not been systematically evaluated.

A wide variety of medications can affect the sleep-wake cycle (Perlis, Smith and Pigeon, 2005). Among the most common medications that can disturb sleep are beta-adrenergic blockers,
thyroid preparations, corticosteroids, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, methyldopa (Aldomet), phenytoin (Dilantin) and some chemotherapeutic agents.

When a medication is prescribed for chronic insomnia, it should be given at the lowest effective dosage and on a short-term basis only. Having the patient take the medication intermittently is best, and discontinuation should be accomplished gradually to avoid rebound insomnia (Sateia, Doghramji and Hauri, 2000).

In an elderly patient, a short-acting benzodiazepine or zolpidem (Ambien) is preferable because these agents reduce the likelihood of residual daytime sedation. Although technically not a benzodiazepine, zolpidem appears to act by binding to benzodiazepine receptors. In general, the maximum benzodiazepine dosage used in an elderly patient should be one half that of the usual adult dosage.

A low dosage of a sedating antidepressant, such as trazodone (Desyrel), has an advantage over traditional hypnotics in that it does not depress respiration, an attribute that could be relevant in patients with sleep apnea. Trazodone is widely used, both alone and as a hypnotic, in patients who develop a sleep disturbance while taking a selective serotonin reuptake inhibitor or a monoamine oxidase inhibitor. However, one serious, though uncommon, adverse effect of trazodone is priapism.

While antihistamines with sedative properties may be beneficial in younger patients, the use of antihistamines such as diphenhydramine (Benadryl) or hydroxyzine (Atarax) is not a good choice in patients because of the potential for anticholinergic side effects such as dizziness and urinary retention. Before sedative medications are prescribed for patients with chronic pain syndromes, it is important to ascertain whether they are receiving adequate pain control.

**Psychological treatment**

Having the patient keep a sleep diary for two weeks may be helpful. Depending on the findings in the sleep diary, a discussion of sleep hygiene may be beneficial. Adopting the practices of good sleep hygiene is often beneficial, whether the patient has primary insomnia or a sleep disturbance related to a medical condition (Walsh & James, 2004). For example, a randomized,
controlled trial demonstrated that moderate-intensity exercise (i.e., low-impact aerobics, brisk walking and stationary cycling) improved self-rated sleep quality in men and women aged 50 to 76 years (Bailey, 1987). Behavioral psychologists focus on encouraging the patient to eliminate behavior incompatible with sleep, such as lying in bed and worrying, by instructing the patient to leave the bedroom at these times. Patients can condition themselves to be insomniacs, and treatment focuses on deconditioning the patient from associating the bedroom with a place of restlessness.

Cognitive/behavioral therapy has been recognized as a valid and successful treatment approach for insomnia. Cognitive/behavioral therapy can include any combination of sleep restriction, sleep hygiene, stimulus control, paradoxical intention, and cognitive restructuring. Many of these commonly used clinical tools have not undergone rigorous testing to determine their efficacy and long-term safety. The efficacy of these treatments has been evaluated in some studies (Zorick et al, 2000; Morin, Colecchi and Stone, 1999), but differences in the definition of insomnia and outcome measures make it difficult to compare study results.

**Treatment Approaches for Chronic Insomnia:**

1. *Conventional Treatment*

The first task is to determine the exact cause of insomnia. In most cases, more than one cause for insomnia is likely. Thus, a careful evaluation and diagnosis are important before strategies for treatment can be determined (Doghramji and Paul, 2004). The underlying cause or causes should be treated if possible. In considering what medication would be appropriate, physicians will consider the patient's age, medical condition, use of alcohol, and need to function when awakened during his or her normal sleep time.

Loney, Chambers and Bennett (1988) studied the socio-demographic profile, symptomatology, prior treatment and treatment response of patients seen in an Insomnia Clinic. The patients in this
sample sought treatment or were referred for treatment much earlier compared to other samples studied. Importantly, 29.5% of the patients referred had an undiagnosed psychiatric condition.

Chronic insomnia requires a thorough physical examination, alteration of some life habits, and perhaps psychotherapy to identify a hidden cause such as Cognitive Behavioral therapy and drug therapy, if necessary, are the preferred approach in this case.

2. Cognitive behavioral therapy (CBT)

Cognitive behavioral therapy has been shown to be a highly effective approach for the treatment of primary insomnia. Findings by Bouchard, Sebastien et al, (2003), suggest that self-efficacy perceptions are useful in predicting adherence to CBT of insomnia. This therapy attempts to alleviate specific behavioral problems by systematically altering the patient’s learned behavior (Montgomery, Perkin & Wise, 1975; Ribordy and Denney, 1977; Bootzin and Nicassio, 1978; Lazarus, 1976; O’Leary and Wilson, 1975). It begins with a comprehensive analysis designed to identify the initial problem and related factors. This analysis is made as much as possible through observation of the patient’s behavior as it occurs naturally. This type of therapy is based on the principle that human behavior is learned and thus can be modified or controlled. First, behavior can be modified by altering the specific conditions that accompany or precipitate it. Second, the type of behavior that occurs in a particular situation can be changed or modified. Third, the consequences of the undesired behavior can be altered, thus modifying future behavior.

Cognitive behavioral therapy for insomnia typically involves:

**Attribution techniques**: a clinical application of attribution theory in terms of source reattribution could involve suggesting to an insomniac patient that his sleep difficulty is related in great part to the patient’s perception that some external factor, such as sleeping pill, is a necessity for sleep (Bootzin, 1976).

**Cognitive restructuring**: cognitive restructuring is an attempt to enable patients to revise their personal beliefs about sleep by correcting misconceptions about basic sleep processes (Goldfried and Goldfried, 1975; Thoresen, Coates et al, 1981). Negative thoughts about sleeplessness are replaced with more positive thoughts.
**Stimulus control**: stimulus control procedures are predicated upon the principle that the sleep of insomniacs is not appropriately associated with environmental stimuli (bed or bedroom), Haynes, Price and Simons (1975). These treatments attempts to eliminate or reduce bedroom activities that are not compatible with sleep and to associate the bed and bedroom only with sleep (Thoresen, Burnett et al, 1980; Bootzin and Nicassio, 1978). A set of instructions aimed at undoing conditioned arousal at bedtime by reassociating the bedroom with rapid sleep onset are given. As insomnia is a significant problem among euthymic patients with bipolar disorder, stimulus control has been found effective.

Sawhney and Chopra, (1986) have used the following instructions.

*Go to bed only when you are sleepy. If you do not fall asleep within 15 minutes or wake up and can't resume sleep within 15 minutes, leave the bedroom and return only when sleepy again. Repeat as often as necessary. Use the bedroom only for sleep and sex. Do not read, watch TV, work, or eat in bed. Get up at the same time every morning, including weekends. Avoid daytime napping.*

**Sleep restriction**: Sleep restriction involves curtailing the amount of time the patient spends in bed to increase the efficiency of sleep. First, restrict the time allowed in bed to equal the average amount of time the patient actually spends sleeping. After each week, the percent of time spent sleeping in bed is calculated. This is called sleep efficiency (SE) index.

Sleep efficiency (SE) index = time spent asleep/time spent in bed x 100

If SE is greater than 85%, an additional 15 to 20 minutes of time in bed is added to the beginning of the night. If SE is less than 85%, time in bed is further restricted by 15 to 20 minutes. Reducing the time in bed to less than 5 hours is not generally recommended. Sleep restriction is very effective if followed closely. Needs discipline on the part of patient for its success.

**Relaxation strategies**: Various relaxation techniques are useful for inducing sleep. One such technique is shavasan, which is a type of yoga helpful in inducing sleep in clients. In this the person is given an appropriate atmosphere to relax and meditate. Shah in 1986 suggested various practices to master the art of sound sleep.
A Preliminary Study with Sleep–Wake Diaries revealed good evidence for cognitive and physiological arousal in chronic insomnia. Accordingly, clinical trial studies of insomnia treatments aimed at reducing arousal, including relaxation and meditation, have reported positive results. Yoga is a multicomponent practice that is also known to be effective in reducing arousal, although it has not been well evaluated as a treatment for insomnia.

Techniques include such methods as hypnosis, progressive relaxation, autogenic training, systematic desensitization, meditation training, and biofeedback (Montgomery, Perkin & Wise, 1975; Ribordy and Denney, 1977; Bootzin and Nicassio, 1978; Lazarus, 1976; O’Leary and Wilson, 1975). These methods are based upon the evidence that insomniacs are tense, anxious, and physiologically aroused at bedtime. The aim of relaxation is to achieve muscle relaxation in order to reduce autonomic parameters, such as heart rate and respiration.

- **Hypnosis**: Early reports suggested some success in improving insomnia by using hypnotically induced relaxation that included post hypnotic suggestion and self hypnosis (Fry, 1963; Hanley, 1965).

- **Progressive relaxation training**: Jacobson developed the technique that teaches the patient to relax systematically. The patient is asked to tense individual muscle groups voluntarily. Muscles then are relaxed gradually while the patient breathes slowly and deeply.

- **Autogenic training**: AT is derived from the observation that hypnosis usually employs suggestions that the subject’s arm is heavy in achieving relaxation (Ribordy and Denney, 1977; Bootzin and Nicassio, 1978; Bootzin, 1976; Schultz and Luthe, 1959).

- **Systematic desensitization**: It requires that insomniac patients first construct a hierarchy of anxiety producing situations. Then, a counter conditioning program is established for each level of the hierarchy. Insomniac patient may be asked to keep a behavioral log, noting the degree of anxiety induced by thoughts about each situation, then to rank the thoughts in order of their increasing anxiety producing potentials (Tan and Pertschuk, 1978).
• **Meditation training:** Various forms of meditation involve focusing attention on a repetitive stimulus or mantra with the aim of inducing inner calmness and tranquility (Woolfolk, 1975), reducing cortical excitation, and lowering metabolic rate.

• **Biofeedback:** In biofeedback, information on physiologic activity of which the patient is usually unaware is transmitted to the patient via electronic instrumentation. There is some evidence that biofeedback may be useful in improving insomnia, although it is not generally used in a private practice setting because of the special equipment required.

**Light phase shift:** Used for insomnia associated with circadian rhythm disturbances. The use of timed exposure to bright light can be very effective in shifting the timing of the major sleep period. Fetveit, Arne et al (2003) added further evidence of the effectiveness of morning bright light exposure in the treatment of disturbed sleep demented nursing home patients. Evening light is indicated if you sleep too early and wake up early (phase advance syndrome) and morning light is used if you sleep late and wake up late (phase delay syndrome). Natural sunlight and bright-light boxes can be used.

**Support, counseling, or psychotherapy:** Patients may require special considerations at work or school. Some may benefit from support groups and/or marriage or family counseling.

The treatment is related to the cause, if the cause can be determined. If there is an obvious physical or psychological disorder causing insomnia, it should be treated. Depression is a very common cause of insomnia and can usually be treated with medication, cognitive behavioral therapy, or interpersonal therapy (Edinger, Wohlgemuth et al, 2003). Attempts to control environmental and lifestyle factors should be made Sleeping drugs should be used only when prescribed by a health care provider.

In a recent study of CBT versus benzodiazepine treatment, CBT was found to be superior at long-term (2 years) follow-up (Karacan, Thornby et al, 1978).
Approximately one third of American adults report that they have sleep problems, and nearly one half of persons with insomnia consider the difficulty to be serious (Kupfer and Reynolds, 1997; Mellinger, Batler and Uhlenhuth, 1985). Not only does insomnia cause daytime drowsiness, it may impair quality of life in other ways; poor sleepers have been found to receive fewer promotions, to have increased rates of absenteeism and to demonstrate poor productivity (Leigh, 1991; Schweitzer, Anglehardt et al, 1992). Also, the risk of motor vehicle crashes is increased in this group because of fatigue (Mitler, Carskadon et al, 1988)

Transient, or intermittent, insomnia lasts only a few days and is usually related to identifiable factors such as acute medical illness, changes in the sleeping environment, self-medication, jet lag and acute or recurring stress from work problems, concerns about health, marital strife, etc. In most cases, this type of insomnia can be relieved with appropriate attention to the inciting stimulus. Chronic insomnia is best defined as the subjective experience of an inadequate quantity or quality of sleep that has persisted for at least one month (American Psychiatry Association, 1994; National Institute of Health, 1984).

Chronic insomnia is more complex than acute transient insomnia, requiring a more directed approach to its identification, etiology and treatment. After establishing the chronicity of the complaint, a differential assessment of chronic insomnia can be made on the basis of whether the patient has difficulty staying asleep as opposed to difficulty falling asleep. Because insomnia is essentially a symptom and not a diagnosis, it is important to try to determine the cause of chronic insomnia and not just reflexively treat the patient with sedative-hypnotic medications. It is important to remember that use of sedative-hypnotics may worsen sleep-disordered breathing such as that associated with obstructive sleep apnea.

**Theoretical perspective**

Chronic insomnia is an altered state of arousal of an individual which may have impact on daily functioning including cognitive decrements. It may be appreciated by comparing with those who have normal sleep patterns. Since chronic insomnia quite often result into its ill effects it becomes a matter of health concern and the persons effected by this problem seek remedy through physicians which after treatments. Chemical as well placebos are common strategies
which seem to partly relieve symptoms yet not fully. Psychological intervention may help chronic insomniacs to manage their problem and to have better functioning and relieve distressing perceptions. Hence, this biphasic study.

The specific problem and its objectives have been placed at the end of next chapter, Review.